In the Specification:

Please amend the specification as shown:

Please delete the paragraph on page 4, lines 5-15, and replace it with the following paragraph:

A1

The invention includes a composition and method of treatment of fungal pathologies of the oral cavity or fungal growth on the surface of dentures. In a preferred embodiment of the invention a therapeutically effective amount of one or more peptides selected from the group of peptides with a C-terminal sequence consisting of KPV **(SEQ ID NO: 1)**, HFRWGKPV **(SEQ ID NO: 3)**, and SYSMEHFRWGKPV **(SEQ ID NO: 4)** used in combination with a therapeutically effective amount of a fungicide selected from the group consisting of: itraconazole, econazole, ketoconazole, miconazole and fluconazole and dissolved into a carrier. More preferably still each peptide has the primary sequence of KPV **(SEQ ID NO: 1)** or VPK-Ac-CC-Ac-**KPV (SEQ ID NO: 8)** (Ac=Acetyl group). Pharmacologically effective concentrations may be as low as 10^{-12} M but may be as high 10^{-4} M. Pharmacologically effective concentration of these peptides may be incorporated into commercial formulations of creams, gels, mouthwashes, toothpastes, tablets, or atomized sprays.

Please delete the paragraph on page 5, lines 3-6, and replace it with the following paragraph:

A2

Figure 1 illustrates the effect of α-MSH (1-13) **(SEQ ID NO: 4)** and (11-13) **(SEQ ID NO: 1)** and the peptide VPK-Ac-CC-Ac-KPV **(SEQ ID NO: 8)** on *C. albicans* colony forming units compared to controls. All three molecules significantly decreased *C. albicans* colony forming units over a broad range of peptide concentrations.

Please delete the paragraph on page 5, lines 7-10, and replace it with the following paragraph:

A3

Figure 2 **(peptides shown in SEQ ID NOS 1, 3, 4, 2 5 and 6, respectively, in order of appearance)** represents a comparison of candidacidal activity of certain melanocortin peptides and

fluconazole (all 10^{-6} M). The most effective of the melanocortin peptides were those including the C-terminal amino acid sequence of α-MSH, namely, α-MSH (1-13) **(SEQ ID NO: 4)**, (6-13) **(SEQ ID NO: 3)** and (11-13) **(SEQ ID NO: 1)**.

Please delete the paragraph on page 5, line 12, and replace it with the following paragraph:

Figure 3C shows the effect of α-MSH (1-13) **(SEQ ID NO: 4)** treatment on germination of *C. albicans*

Please delete the paragraph on page 5, line 13, and replace it with the following paragraph:

Figure 3D shows the effect of α-MSH (11-13) **(SEQ ID NO: 1)** treatment on germination of *C. albicans*

Please delete the paragraph on page 5, lines 16-18, and replace it with the following paragraph:

Figure 4 illustrates the effect of α-MSH (1-13) **(SEQ ID NO: 4)** and (11-13) **(SEQ ID NO: 1)** on *C. albicans* killing by human neutrophils. Values are expressed as percent increase in killing vs. medium along. Scores are means ± SEM.

Please delete the paragraph on page 5, lines 19-20, and replace it with the following paragraph:

Figure 5 illustrates the effect of α-MSH (1-13) **(SEQ ID NO: 4)**, (11-13) **(SEQ ID NO:1)**, and forskolin on cAMP content of *C. albicans*.

Please delete the paragraph on page 5, lines 21-22, and replace it with the following paragraph:

A8

Figure 6 illustrates the inhibitory effect of α-MSH (1-13) **(SEQ ID NO: 4)**, (11-13) **(SEQ ID NO: 1)**, and forskolin on *C. albicans* colony forming units.

Please delete the paragraph on page 6, lines 3-18, and replace it with the following paragraph:

A9

α-MSH is a 13 amino acid **(SEQ ID NO: 4)**, fungicidal peptide with the primary sequence SYSMEHFRWGKPV **(SEQ ID NO: 4)**. In addition to its fungicidal properties it also anti-pyretic and anti-inflammatory. The C-terminal trimer, KPV **(SEQ ID NO: 1)**, appears responsible for these properties. Lipton, J.M., Antipyretic and Anti-inflammatory Lys-Pro-Val **(SEQ ID NO: 1)** Compositions and Methods of Use, U.S. Patent No. 5,028,592, issued July 2, 1991; Lipton, J.M., Antipyretic and Anti-inflammatory Lys-Pro-Val **(SEQ ID NO: 1)** Compositions and Methods of Use, U.S. Patent No. 5,157,023, issued October 20, 1992; Catania, A., Lipton J.M., α-Melanocyte Stimulating Hormone in the Modulation of Host Reactions, *Endocr. Rev.* 14, 564-576 (1993); Lipton, J. M., Catania, A., Anti-inflammatory Influence of the Neuroimmunomodulator α-MSH, *Immunol. Today* 18, 140-145 (1997). herein incorporated by reference. All references are hereby incorporated by reference in their entirety. The core α-MSH sequence (4-10) **(SEQ ID NO: 2)** has learning, memory and behavioral effects but little antipyretic and anti-inflammatory activity. Lipton, J.M., Catania, A., Anti-inflammatory Influence of the Neuroimmunomodulator α-MSH, *Immunol. Today* 18, 140-145 (1997). α-MSH , the α-MSH core and its tripeptide C-terminal have very low toxicity. Lipton, J.M., Catania, A., Anti-inflammatory Influence of the Neuroimmunomodulator α-MSH, *Immunol. Today* ~~18~~18, 140-145 (1997).

Please delete the paragraph on page 6, line 19 to page 7, line 2, and replace it with the following paragraph:

A10

α-MSH is produced by the post translational processing of propriomelanocortin and shares the 1-13 primary sequence with adrenocortitrophic hormone (ACTH). Eberle, A. N., The Melanotropins, Karger, Basel, Switzerland (1988). It is secreted by a wide variety of cell types,

Cont A10

including pituitary cells, monocytes, melanocytes, keratinocytes, epidermal cells and the epithelial cells of mucous membranes. Lipton, J.M., Catania, A., Anti-inflammatory Influence of the Neuroimmunomodulator α-MSH, *Immunol. Today* 18~~18~~, 140-145 (1997); see also Catania et al., unpublished.

Please delete the paragraph on page 7, lines 3-16, and replace it with the following paragraph:

α-MSH reduces inflammation and fever by modulating the inflammatory cascade locally and systemically. Rajora, N., Ceriani, G., Catania, A., Star, R.A., Murphy, M.T., Lipton, J.M., α-MSH Production, Receptors and Influence of Neopterin, in a Human Monocyte/macrophage Cell Line, *H.Leukoc. Biol.* 59, 248-253 (1996); Star, R.A., Rajora, N. Huang, J., Stock, R.C.,Catania, A., Lipton, J.M., Evidence of Autocrine Modulation of Macrophage Nitric Oxide Synthase by α-MSH, *Proc. Natl. Acad. Sci.* 92~~92~~, 8016-8020 (1995); Lipton, J.M., Ceriani, G., Macaluso, A., McCoy, D., Carnes, K., Biltz, J., Catania, A., Anti-inflammatory Effects of the Neuropeptide α-MSH in Acute, Chronic and Systemic Inflammation, *Ann. N.Y. Acad. Sci.* 741~~741~~, 137-148 (1994); Rajora, N., Boccoli, G., Burns, D., Sharma, S., Catania, A., Lipton, J.M., α-MSH Modulates Local Circulating Tumor Necrosis Factor A in Experimental Brain Inflammation, *J. Neurosci*, 17, 2181-2186 (1997); Richards, D.B., *Lipton, J.M. Effect of* α-MSH (11-13) (Lys-Pro-Val) **(SEQ ID NO: 1)** on Fever in Rabbits, *Peptides* 5~~5~~, 815-817 (1984); Hiltz, M.E., Lipton, J.M., Anti-inflammatory Activity of a COOH-terminal Fragment of the Neuropeptide α-MSH, FASEB J. 3~~3~~, 2282-2284 (1989).

A11

Please delete the paragraph on page 7, line 17 to page 8, line 3, and replace it with the following paragraph:

The broadest aspect of the invention is a composition and method of treatment of fungal pathologies of the oral cavity or fungal growth on the surface of dentures. In a preferred embodiment of the invention a therapeutically effective amount of one or more peptides selected from the group of peptides with a C-terminal sequence consisting of KPV **(SEQ ID NO: 1)**, HFRWGKPV **(SEQ ID NO: 3)**, and SYSMEHFRWGKPV **(SEQ ID NO: 4)** is incorporated into a carrier. More preferably still, each peptide has the primary sequence of KPV **(SEQ ID NO: 1)** or VPK-Ac-CC-Ac-KPV **(SEQ ID NO: 8)** (Ac=Acetyl group). Pharmacologically effective concentrations may be as low as 10^{-12} M but may be as high as 10^{-4} M. A preferred embodiment of the invention utilizes peptide

A12

Cont 12

concentrations of 10^{-12} M to 10^{-10} M. Pharmacologically effective concentrations of these peptides may be incorporated into commercial formulations of creams, gels, mouthwashes, toothpastes, tablets, or atomized sprays.

Please delete the paragraph on page 8, lines 7-10, and replace it with the following paragraph:

A13

Set forth below are examples of various formulations of the invention. As used below the term "Active ingredient" refers to one or more peptides selected from the group of peptides with a C-terminal sequence consisting of KPV **(SEQ ID NO: 1)**, HRFWGKPV **(SEQ ID NO: 3)** and SYSMEHFRWGKPV **(SEQ ID NO: 4)**. Preferably, the active ingredient is KPV **(SEQ ID NO: 1)** or VPK-Ac-CC-Ac-KPV **(SEQ ID NO: 8)**.

Please delete the paragraph on page 10, lines 1-10, and replace it with the following paragraph:

A14

An exemplary formulation of a mouthwash comprises:

Water	89.0g
1-methoxypropanol	7.0g
n-propanol	1.0g
Saccharine	.06g
Glycerol	1.3g
Flavoring	1.0g
VPK-Ac-CC-Ac-KPV dimer **(SEQ ID NO: 8)**	.1-.1*10^{-8} mg

Please delete the paragraph on page 11, line 26 to page 12, line 4 and replace it with the following paragraph:

A15

The peptides used in the following examples include: α-MSH (1-13) (SEQ. ID. NO. 4), (4-10) (SEQ. ID. **NO:** ~~002~~) (6-13) (SEQ. ID. **NO:** ~~003~~), and (11-13) (SEQ. ID. NO. 1), all of which were N-acetylated and C-amidated, and ACTH (1-39) **(SEQ ID NO: 5)** and (18-39) **(SEQ ID NO: 6)**

Cont A15

(CLIP). These peptides were prepared by solid-phase peptide synthesis and purified by reversed phased high performance liquid chromatography. Some examples also include a dimer of the amino acid sequence KPV (SEQ. ID. NO. 1), VPK-Ac-CC-Ac-KPV (SEQ. ID. NO. 8), which also was N-acetylated and C-amidated (KPV dimer) **(SEQ ID NO: 1)**. Dimers can be formed by adding cysteines at the N-termini of any of the above polypeptides and allowing the cysteines of two polypeptides to form a disulfide bond. Both homo-dimers and hetero-dimers can be formed using this method.

Please delete the paragraph on page 12, lines 16-18, and replace it with the following paragraph:

EXAMPLE 1

A16 The first example suggests that α-MSH (11-13) **(SEQ ID NO: 1)**, (6-13) **(SEQ ID NO: 3)** and (1-13) **(SEQ ID NO: 4)** exhibit similar anticandidal properties as flucanazole over an exceedingly broad range of concentrations.

Please delete the paragraph on page 12, lines 19-23, and replace it with the following paragraph:

A17 *C. albicans* ($1x10^6$/ml in HBSS) was incubated in the presence of absence or (1-13) **(SEQ ID NO: 3)** or (11-13) **(SEQ ID NO: 1)** at concentrations in the range of 10^{-15} M to 10^{-6} M for 2 hours at 37° C. Cells were then washed in cold distilled water and diluted with HBSS to a concentration of 100 organisms/ml. One-ml aliquots were dispensed on blood agar plates and incubated for 48 hours at 37° C. Organism viability was estimated from the number of colonies formed.

Please delete the paragraph on page 13, lines 1-4, and replace it with the following paragraph:

A18 In subsequent experiments using familiar procedures we compared activity of α-MSH (4-10) **(SEQ ID NO: 2)**, (6-13) **(SEQ ID NO: 3)**, (11-13) **(SEQ ID NO: 1)**, ACTH **(SEQ ID NO: 5)** (1-39), (18-39) **(SEQ ID NO: 6)** and fluconazole, the latter an established antifungal agent.

Cont A18

Melanocortin peptides and fluconazole were tested in concentrations of 10^{-6} M to 10^{-4} M. There were at least six replicates for each concentration of peptide.

Please delete the paragraph on page 13, lines 5-8, and replace it with the following paragraph:

A19

Fig. 1 shows that *C. albicans* colony forming units (CFU) were greatly reduced by α-MSH (1-13) **(SEQ ID NO: 4)** and (11-13) **(SEQ ID NO: 1)**. Fig. 1 also shows that the VPK-Ac-CC-Ac-KPV **(SEQ ID NO: 8)** peptide also inhibited *C. albicans* colony formation). Concentrations of all three peptides from 10^{-12} M to 10^{-4} M had significant inhibitory effects on CFU ($p<.01$ vs. control).

Please delete the paragraph on page 13, lines 9-18, and replace it with the following paragraph:

A20

Fig. 2 demonstrates that in experiments comparing the relative potency of 10^{-4}M melanocortin peptides in reducing *C. albicans* viability, α-MSH (11-13) **(SEQ ID NO: 1)**, (6-13) **(SEQ ID NO: 3)** and (1-13) **(SEQ ID NO: 4)** were the most effective. Their inhibitory activity was similar to that of equimolar fluconazole. The core α-MSH sequence (4-10) **(SEQ ID NO: 2)** which has behavioral effects but little anti-inflammatory activity, caused approximately 50% inhibition of CFU. Fig. 2 also shows that although this inhibitory effect was substantial ($p<.01$ vs. control), it was significantly less than that caused by α-MSH fragments bearing the KPV **(SEQ ID NO: 1)** signal sequence, i.e., α-MSH (6-13) **(SEQ ID NO: 3)** and (11-13) **(SEQ ID NO: 1)** $p<.01$), or the parent molecule α-MSH(1-13) **(SEQ ID NO: 4)** ($p<.05$). ACTH (1-39) **(SEQ ID NO: 5)** and the ACTH fragment (18-39) **(SEQ ID NO: 6)** did not reduce *C. albicans* viability. Even higher concentrations of these ACTH peptides (up to 10^{-4} M) were likewise ineffective in reducing *C. albicans* CFU (results not shown in the figures).

Please delete the paragraph on page 13, line 19 to page 14, line 9, and replace it with the following paragraph:

A21

These results show that α-MSH (1-13) **(SEQ ID NO: 4)**, its C-terminal tripeptide (11-13) **(SEQ ID NO: 1)**, and other α-MSH fragments have significant fungicidal effects against *C. albicans*. The most effective of the α-MSH peptides were those including the C-terminal amino acid sequence

KPV **(SEQ ID NO: 1)** of the α-MSH sequence, i.e., α-MSH (1-13) **(SEQ ID NO: 4)**, (6-13) **(SEQ ID NO: 3)** and (11-13) **(SEQ ID NO: 1)**. In addition, the sequence VPK-Ac-CC-Ac-KPV **(SEQ ID NO: 8)** has also been shown to be at least as effective α-MSH (11-13) **(SEQ ID NO: 1)** against microbes. The α-MSH cores sequence (4-10) **(SEQ ID NO: 2)**, which is known to influence learning and memory, but has little antipyretic and anti-inflammatory influence, was effective, but less so. The ACTH peptides (1-39) **(SEQ ID NO: 5)** and (18-39) **(SEQ ID NO: 6)** did not have significant candidacidal effects. These observations indicate that antifungal activity is not common to all melanocortin peptides, but rather that is specific to α-MSH amino acids sequences, and most particularly to the C-terminal amino-acid sequences of α-MSH. This strongly suggest that α-MSH(1-13) **(SEQ ID NO: 4)**, its C-terminal tripeptide (11-13) **(SEQ ID NO: 1)**, and other α-MSH fragments could server as a basis for a therapeutic treatment of acute and chronic candidal infections of the oral cavity or as antifungal agent against cadidal growth on denture surfaces.

Please delete the paragraph on page 14, lines 11-21, and replace it with the following paragraph:

EXAMPLE 2

Example 2 demonstrates that α-MSH (1-13) **(SEQ ID NO: 4)**, (6-13) **(SEQ ID NO: 3)** or (11-13) **(SEQ ID NO: 1)** strongly inhibits Candidal germination. *C. albicans* from stationary phase cultures were washed twice with distilled water and suspended in HBSS to a final concentration of 2 x 10^6/ml. Hyphal growth was induced by addition of 10% inactivated horse serum (GIBCO/BRL, Great Britain) to yeast incubated for 45 minutes at 37° C with continuous shaking. Horse serum was removed by washing cells twice with HBSS and incubation was continued for 60 minutes at 37°C in the presence of α-MSH (1-13) **(SEQ ID NO: 4)**, (6-13) **(SEQ ID NO: 3)** or (11-13) **(SEQ ID NO: 1)** at a concentration of 10^{-6} M with continuous shaking. The percentage of filamentous cells was evaluated under a light microscope with the aid of hemocytometer. Experiments were run in triplicate and at least 200 cells were scored. Photomicrophraphs were taken with a MC100 camera attached to an Axioskop Zeiss microscope.

Please delete the paragraph on page 14, line 22 to page 15, line 2, and replace it with the following paragraph:

A23

Figs. 3A-D show that coincubation of *C. albicans* with α-MSH (1-13) **(SEQ ID NO: 4)** or (11-13) **(SEQ ID NO: 1)** inhibited germ tube formation induced by horse serum, α-MSH (1-13) **(SEQ ID NO: 4)** caused 28-32% reduction in the number of filamentous cells; the tripeptide inhibited germination by 54-58%. The octapeptide α-MSH (6-13) **(SEQ ID NO: 3)** had similar activity (approximately 50% inhibition)(not shown).

Please delete the paragraph on page 15, lines 3-13, and replace it with the following paragraph:

A24

The pathogenesis of *C. albicans* infection involves adhesion of yeast cells to epithelial cells, commonly found in the mucosal membranes of the ears, eyes, nose and throat and/or endothelial cells, followed by morphologic switching of the yeast cells from the ellipsoid blastospore to various filamentous forms: germ tubes, pseudohyphae and hyphae. Gow, N.A., Germ Tube Growth of Candida Albicans, *Curr. Topics Med. Mycol.* 8, 43-45 (1997). The results also show that in addition to direct candicidal properties, α-MSH(1-13) **(SEQ ID NO: 4)**, its C-terminal tripeptide (11-13) **(SEQ ID NO: 1)**, and other α-MSH fragments interfere with germination and adhesion of candida to the epithelium. This suggests that if the germination and adhesion of candida could be interfered with, the invasive forms of chronic candidiasis that innervate the epithelium could be treated with therapy based upon α-MSH(1-13) **(SEQ ID NO: 4)**, its C-terminal tripeptide (11-13) **(SEQ ID NO: 1)**, and other α-MSH fragments.

Please delete the paragraph on page 16, lines 1-10 and replace it with the following paragraph:

A25

C. albicans (1×10^6) were opsonized with human AB serum in a shaking water bath for 30 minutes at 37°C. Organisms were then incubated with neutrophils in medium or in medium with α-MSH (1-13) **(SEQ ID NO: 4)** or α-MSH(11-13) **(SEQ ID NO: 1)** in concentrations of 10^{-15}M to 10^{-4} M in a shaking water bath for 2 hours at 37°C. After incubation, the culture tubes were placed on ice stop growth and extracellular organisms were washed twice with centrifugation at 1000 x g at 4°C. A

2.5% sodium desoxycholoate solution was added to obtain a suspension of 10^6 cells/ml. Two 1/100 serial dilutions in HBSS were made to obtain a final suspension of 100 cells/ml. Aliquots of 1 ml were dispensed on blood agar plates and incubated for 48 hours at 37°C. Colony forming units (CFUs) were counted at the end of the incubation period. Experiments were run in triplicate and repeated using blood from 5 different donors.

Please delete the paragraph on page 16, lines 11-16 and replace it with the following paragraph:

Fig. 4 shows that α-MSH(1-13) **(SEQ ID NO: 4)** and (11-13) **(SEQ ID NO: 1)** enhanced the killing of *C. albicans* by human neutrophils when administered in concentrations of 10^{-12} M to 10^{-4} M ($p<.01$). Therefore, enhanced killing occurred over a very broad range of concentrations including picomolar concentrations, i.e. the quantity of α-MSH found in human placenta. Catania, A., Airaghi, L., Garofalo, L., Cutuli, M., Lipton, J.M., The Neuropeptide α-MSH in AIDS and Other Conditions in Humans, *Ann. N.Y. Acad. Sci.* 840~~840~~, 848-856 (1998).

Please delete the paragraph on page 16, lines 17-23 and replace it with the following paragraph:

Reduced killing of pathogens is a dire consequence of therapy with corticosteroids and nonsteroidal anti-inflammatory drugs during infection. Stevens, D.L., Could Nonsteroidal Anti-inflammatory Drugs (NSAIDS) Enhance Progression of Bacterial Infections to Toxic Shock Syndrome?, *Clin. Infect. Dis.*, 21~~21~~, 977-80 (1997); Capsoni, F., Meroni, P.L., Zocchi, M.R.,Plebani, A.M., Vezio, M., Effect of Corticosteroids on Neutrophil Function: Inhibition of Antibody-dependent Cell-mediated Cytotoxicity (ADCC), *J Immunolpharmacol.* 5~~5~~ 217-230 (1983). This effect is particularly dangerous in immunocompromised patients.

Please delete the paragraph on page 17, lines 1-4 and replace it with the following paragraph:

These results also suggest that α-MSH(1-13) **(SEQ ID NO: 4)**, its C-terminal tripeptide (11-13) **(SEQ ID NO: 1)**, and other α-MSH fragments would be useful for treatment of candidiasis in

Cont A28

immunocompromised patients since these peptides appear not to reduce neutrophil chemotaxis and thus would not further comprise the immune system.

Please delete the paragraph on page 17, lines 6-15 and replace it with the following paragraph:

A29

EXAMPLE 4

Example 4 suggests a cellular mechanism to explain how α-MSH exerts its anti-candidal properties. *C .albicans* (10^6/ml), permeabilized with toluene/ethanol, were incubated at 37°C with continuous shaking in the presence of 10^{-6} M α-MSH(1-13) **(SEQ ID NO: 4)**, (11-13) **(SEQ ID NO: 1)**, forskolin, an agent known to increase intracellular cAMP, or in medium alone. The reaction was stopped after 3 minutes by the addition of ice cold ethanol, cAMP was measured in duplicate using a commercial enzyme immunoassay (EIA) kit (Amersham, United Kingdom) after extraction via the liquid-phase method according to manufacturer's instructions. The effect of forskolin (10^{-6} M) on *C. albicans* colony formation was determined using the same procedure as for α-MSH peptides.

Please delete the paragraph on page 17, lines 16-23 and replace it with the following paragraph:

A30

Because many of the effects of α-MSH are known to be mediated by induction of cAMP, we measured effects of α-MSH peptides on cAMP accumulation in *C. albicans.* Fig.5 shows that α-MSH (1-13) **(SEQ ID NO: 4)** and (11-13) **(SEQ ID NO: 1)** enhanced cAMP content in the yeast. Fig. 6 shows the increase was of the same order of magnitude as the induced by equimolar forskolin, an adenylate cyclase activator. To determine whether increases in cAMP could be responsible for reduction in CFU, we tested the effects of forskolin on *C. albicans* viability. Results showed that 10^{-6}M forskolin markedly inhibited *C. albicans* CFU relative to control ($p<.01$). Fig. 6 demonstrates that the inhibitory effect was similar to that exerted by α-MSH.

Please delete the paragraph on page 18, lines 1-8 and replace it with the following paragraph:

A31

The mechanism of action of natural antimicrobial agents is only partly understood. Most of these peptides, including the defensins, alter membrane permeability and impair internal homeostasis

Cont A31

of the organism. The first contact is made between the cationic groups of the peptide and the negatively charged head of the target membrane. Then, the tertiary structure determines the mode of insertion of the peptide into membranes where they from ion channels or pores that disrupt cell integrity. It is known that cAMP-enhancing agents inhibit mRNA and protein synthesis in *C. albicans*. Bhattacharya, A., Datta, A., Effect of Cyclic AMP on RMA and Protein Synthesis in *C. albicans, Biochem. Biophys. Res. Commun.* 7777: 1483-44 (1977).

Please delete the paragraph on page 18, line 18 to page 19, line 17 and replace it with the following paragraph:

A32

Although the specific amino acid sequence described here are effective, it is clear to those familiar with the art that amino acids can be substituted in the amino acid sequence or deleted without altering the effectiveness of the peptides. Further, it is known that stabilization of a the α-MSH sequence can greatly increase the activity of the peptide and that substitution of amino acid D-forms for L-forms can improve or decrease the effectiveness of peptides. For example, a stable analog of α-MSH, [Nle^4,$D\text{-}Phe^7$]-α-MSH **(SEQ ID NO: 10)** which known to have marked biological activity on melanocytes and melanoma cells, is approximately 10 times more potent than the parent peptide in reducing fever. Holdeman, M., and Lipton, J.M., Antipyretic Activity of a Potent α-MSH Analog, *Peptides* 6, 273-5 (1985). Further, adding amino acids to the C-terminal α-MSH (11-13) **(SEQ ID NO: 1)** sequence can reduce or enhance antipyretic potency. Deeter, L.B., Martin, L.W., Lipton, J.M., Antipyretic Properties of Centrally Administered α-MSH Fragments in the Rabbit, *Peptides* 9, 1285-8 (1989). Addition of glycine to form the 10-13 sequence slightly decreased the potency; the 9-13 sequence was almost devoid of activity, whereas the potency of the 8-13 sequence was greater than that of the 11-13 sequence. It is known that Ac-[$D\text{-}K^{11}$]-α-MSH 11-13-NH_2 **(SEQ ID NO: 11)** has the same general potency as the L-form of the tripeptide α-MSH 11-13 **(SEQ ID NO: 1)**. Hiltz, M.E., Catania, A., Lipton, J.M., Anti-inflammatory of α-MSH (11-13) **(SEQ ID NO: 1)** Analogs; Influences of Alterations in Stereochemistry, *Peptides* 12, 767-71 (1991). However, substitution with D-proline in position 12 of the tripeptides rendered it inactive. Substitution with the D-form of valine position 13 or with the D-form of lysine at position 11 plus the D-form of valine at position 13 resulted in greater anti-inflammatory activity than with the L-form tripeptide. These examples indicate that alteration in the amino acid characteristics of the peptides can influence activity of the peptides or have little effect, depending upon the nature of the manipulation.

Please delete the Abstract and replace it with the following Abstract:

ABSTRACT OF THE INVENTION

A33

The broadest aspect of the invention is a composition and method of treatment of fungal pathologies of the oral cavity or fungal growth on the surface of dentures. A preferred embodiment of the invention is a pharmacologically effective amount of a peptide selected from the group of peptides with a C-terminal sequence consisting of KPV **(SEQ ID NO: 1)**, HFRWGKPV **(SEQ ID NO: 3)**, and SYSMEHFRWGKPV **(SEQ ID NO: 4)** in combination with a therapeutically effective amount of a fungicide selected from the group consisting of: itraconazole, econazole, ketoconazole, miconazole, imconazole and fluconazole. Another embodiment of the invention is a method for treating fungal pathologies of the ~~of~~ oral cavity and dentures by application of a pharmacologically effective amount of a peptide selected from the group of peptides with a C-terminal sequence consisting of KPV **(SEQ ID NO: 1)**, HFRWGKPV **(SEQ ID NO: 3)**, and SYSMEHFRWGKPV **(SEQ ID NO: 4)** in combination with a therapeutically effective amount of a fungicide selected from the group consisting of: itraconazole, econazole, ketoconazole, miconazole and fluconazole. In yet another embodiment of the invention these peptides are used in combination with a therapeutically effective amount of gram positive and/or gram negative antibiotics.